Corrigendum

Corrigendum to: Depolarisation of the plasma membrane in the arsenic trioxide (As2O3)- and anti-CD95-induced apoptosis in myeloid cells (FEBS 29005) [FEBS Letters 578 (2004) 85–89]☆

Florian Nolte[a], Oliver Friedrich[b], Markus Rojewski[a], Rainer H.A. Fink[b], Hubert Schrezenmeier[a], Sixten Körper[a,*]

[a] *Department of Transfusion Medicine, University of Ulm, Institute for Clinical Transfusion Medicine and Immunogenetics, Ulm, Germany*
[b] *Medical Biophysics, Institute of Physiology and Pathophysiology, University of Heidelberg, Heidelberg, Germany*

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We recently found out that in some of our experiments a Ringer solution may have been used with an accidentally increased Na^+-concentration (about 1.5 times too high). We therefore repeated all experiments in which Ringer solution was used with the solutions now containing correct [Na^+]. From these experiments, some data of the figures have to be corrected. In Fig. 1, the calibration with 1 nM gramicidine is now shifted by 7.8 mV towards more depolarised values. Accordingly, the potentials at 10 and 20 μM As_2O_3 and anti-Fas now read: −26.8, −6.9 and −45.3 mV. The choline-Cl solution (Fig. 3) shows a more pronounced inhibition of apoptotic depolarisation after 3 h of incubation with anti-Fas (from 16.2 ± 3.4% depolarised cells to 1.4 ± 1.0%, $P = 0.002$) and after 24 h incubation with As_2O_3 (from 49.9 ± 4.9% to 38.6 ± 5.1%, $P = 0.012$). Finally, in Fig. 4C, amiloride inhibits apoptotic depolarisation from 13.4 ± 1.3 % to 9.7 ± 1.6%. Thus, it can additionally be concluded that: (i) a Na^+-influx may contribute to early stages of anti-Fas and late stages of As_2O_3 induced apoptotic depolarisations; and (ii) the ENaC-like channel – in contrast to our previous view may contribute to the apoptotic depolarisation.

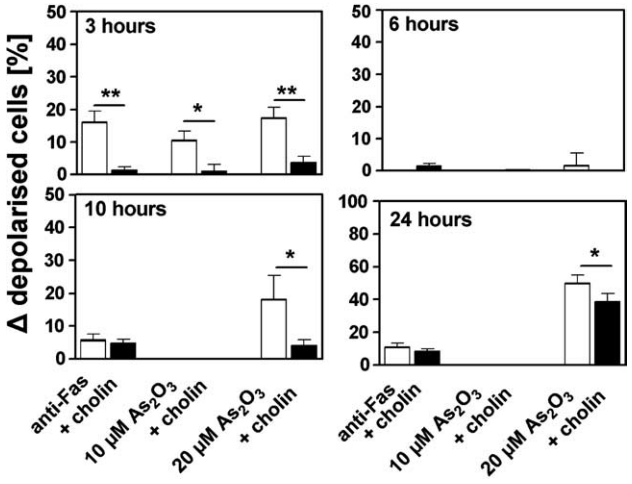

Fig. 3.

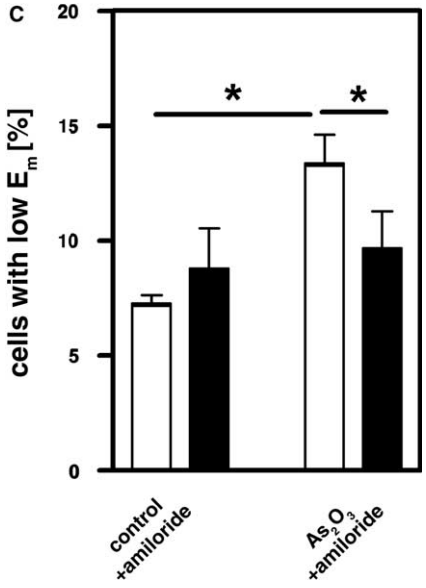

Fig. 4C.

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*Corresponding author. Fax: +49 731 150500.
E-mail address: sixten.koerper@medizin.uni-ulm.de (S. Körper).

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